THE TAMILNADU

Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI

A STUDY OF PANCREATIC DISEASES

DISSERTATION SUBMITTED FOR M.S. DEGREE BRANCH 1

(GENERAL SURGERY)

FEBRUARY – 2006

DEPARTMENT OF GENERAL SURGERY
MADURAI MEDICAL COLLEGE
MADURAI.

CERTIFICATE

This is to certify that the dissertation entitled **"Study of Pancreatic Diseases"** is a bonafide record of work done by **Dr. A. Sureshkumar,** in the Department of Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai.

Prof. Dr. K.V.Maheswaran, M.S.,
Reader in Surgery
Govt. Rajaji Hospital and
Madurai Medical College
Madurai

Prof. Dr. M. Kalyanasundaram, FICS., M.S.,
Professor and H.O.D of Surgery
Govt. Rajaji Hospital and
Madurai Medical College
Madurai

ACKNOWLEDGEMENT

I wish to express my sincere gratitude to my Chief **Prof.K.V.MAHESWARAN, MS.,** *Reader in Surgery* for having helped me at every step in successfully completing this study.

I wish to express my sincere thanks to **Prof.M.KALYANASUNDARAM, MS.,** *Professor and Head of the Department of Surgery* for his sincere advice and co-operation for completing this study.

I also thank my *Professor and Head of the Department of Pathology* and to all teachers who helped me to complete this study in time.

I am also very grateful to our *Dean* **Dr.M.VASANTHA,MD.,** Her kind permission to utilize the clinical material for this study, in time.

I also extend my thanks to all my patients for their full co-operation in successfully conducting this study.

TABLE OF CONTENTS

INTRODUCTION

The pancreas cuddles the let kidney, tickles the spleen, hugs the duodenum, cradles the aorta, opposes the inferior vena cava, dallies with the right renal pedicle, hides behind the posterior parietal peritoneum of the lesser sac and wraps itself around the superior mesenteric vessels.

Historically, the first description of the pancreas is credited to Herophilus of Chaikaldon around the year 300 B.C. Four centuries later in approximately 100 A.D this abdominal organ was named the pancreas by Rufus of Ephesus. The first operative intervention on the pancreas has been attributed to Le Dentu in the year 1862, involving percutaneous aspiration of a pancreatic mass with an unfavourable outcome. The first successful resection of a periampullary tumour was performed by Halsted in 1899. The tumour was resected locally and reconstruction was performed. The first successful pancreatico duodenectomy was performed by kausch in 1912. In 1935, Whipple done a successful two stage enbloc resection of the head of the pancreas and the duodenum. The first one stage pancreatico duodenectomy was reported in the United States by Trimble in 1941.

Surgeries in pancreas were considered at one point of time equivalent to disturbing a sleeping tiger. But the scenario today is entirely different.

The better understanding of anatomy and modern investigation have made surgeries of pancreas a child’s play under competent hands.

Even through the incidence of pancreatic diseases are not uncommon in this surgical side, the morbidity and mortality is relatively high when compared with any other organic diseases.

Sine the incidence of alcoholism is increasing, the disease of pancreas is also expected to be more prevalent. Hence we decided to study this topic in detail in various aspects such as in incidence; surgeries and various modalities of treatment.

AIM OF STUDY

The aim of this study was to evaluate the etiopathogeneis of various pancreatic pathologies ranging from inflammatory lesions to malignancies and catogorise the various modalities of treatment and their outcome, so that a better prognosis is afforded to our patients in our hospital

MATERIALS AND METHODS

This study consists of all patients admitted in surgical and Gastro enterology ward of Govt. Rajaji Hospital, Madurai from Feb 2003 to August.2005. Once the patient is admitted the name, age, sex were noted. Clinical evaluation was done in a systematic way Appropriate investigations were carried out. In those who were operated, the operative findings & methods of management are recorded. Cases were followed up till their discharge from the hospital. Above facts were recorded in a proforma prepared for this study. Patient reviewed at regular intervals.

ANATOMY

A revision of the surgical anatomy of the pancreas is necessary at this juncture to appreciate the various aspects of surgeries of pancreas. Pancreas lies transversely across the upper part of the posterior abdominal wall and is about 15-20cm in length. 3.1 cm in width; 1-1.5cm in thickness and weighs about 80-90 gms.

Posterior to the pancreas is the inferior vena cava, Aorta, Lt Kidney, both renal veins and right renal artery. Pancreatic head lies with in the concave sweep of the duodenum. Body crosses the spine and is directed some what obliquely and superiorly to the left with the tail residing in the hilum of the spleen. Splenic artery runs along the upper border of the pancreas, and the splenic vein runs behind, just superior to the lower edge. The superior mesenteric vein and artery lies just behind the neck of pancreas and are also enclosed posteriorly by an extension of the head known as uncinate process. The uncinate process lies between the inferior vena cava and the portal vein.

The main pancreatic duct of Wirsung usually traverses the entire length of the gland slightly above a line halfway between the superior and inferior edges and normally ends by joining the common bile duct. The accessory duct of

Santorini branches out from the pancreatic duct in the neck of the pancreas and empties into the duodenum, about 2.5cm above the duodenal papillae.

THE ARTERIAL AND VENOUS BLOOD SUPPLY

Head and neck of pancreas are supplied by branches from the anterior and posterior pancreatico – duodenal arcades. Which are “formed by the union of superior and inferior pancreatico – duodenal arteries. Superior pancreatico duodenal artery is a branch of gastro-duodenal trunk which itself is a branch of right hepatic artery. The inferior pancreatico – duodenal artery is a branch from the superior mesenteric artery.

The body and tail of pancreas are supplied by a number of branches arising from the splenic artery viz dorsal pancreatic artery, inferior pancreatic artery, arteria pancratica magna and caudal pancreatic arteries.

VENOUS DRAINAGE

Is into the portal, spleenic and superior mesenteric vein.

LYMPAHATIC DRAINAGE

Lymph capillaries commence around the acini and their continuations following the blood vessels. There are no lymphatics in the pancreatic islets.

Most vessels end in the pancreatico-spleenic nodes. Some in nodes along the pancreatico-duodenal vessels, and others in the superior mesenteric pre-aortic nodes.

SURGICAL PHYSIOLOGY OF PANCREAS

In response to a meal, pancreas secrete digestive enzymes in an alkaline (P.H.8,4) bicarbonate rich fluid. Acinar cells synthesize and secrete digestive

enzymes, while the duct cells secrete bicarbonate. Daily secretion is about one litre containing 5-8gm of protein in the form of enzymes. About 20 digestive enzymes are produced.

1. Proteolytic enzymes-eg trypsin
2. Lipolytic enzymes-eg lipase
3. Srtach splitting enzymes –eg. Amylas
4. Nucleci acid splitting enzymes –eg ribonuclease

Pancreatic enzyme secretion is potently stimulated by Pancreozymin which is released from the duodenal mucosa by luminal fat and peptides.

Rate of secretions and the bicarbonate content of the pancreatic juice is increased by hormone SECRETIN which is released from the duodenal mucosa by luminal acid stimulation.

Pancreatic secretion can also be stimulated by hormones produced with in the pancreas and small bowel, vasoactive intestinal peptide, (VIP) and gastrin and inhibited by the pancreatic hormones, somatostatin, pancreatic polypeptide and glucagons.

Pancreatic secretions consists of a CEPHALIC PHASE- initiated by thought of food, a GASTIC PHASE produced by food in the stomach and an INTESTIANL PHASE mediated by secretion of pancreozymin released from the duodenum and jejunum.

PATHOLOGY OF PANCREATIC DISEASES

The most significant disorders of the exocrine pancreas are cystic fibrosis, acute and chronic pancreatitis and tumours.

In cystic fibrosis, pancreatic abnormalities are present in approximately 85 to 90% of patients. In milder cases there may be only accumulations of mucous in the small ducts with some dilatation of the exocrine glands. In more advanced cases, usually seen in older children or adolescents, the ducts are totally plugged, causing atrophy of the exocrine glands and progressive fibrosis.

In acute pancreatitis four basic alterations are seen histologically. 1.Proteolytic destruction of pancreatic substance, 2.necrosis of blood vessels with subsequent haemorrhage, 3.necrosis of fat by lipolytic enzymes and 4.an accompanying inflammatory reaction. These alterations depends on the duration and severity of the diseases. Acute pancreatic necrotic lesions consists of enzymatic destruction of fat cells in which the vacuolated fat cells are transformed to shadowy outlines of cell membranes filled with pink granular, opaque precipitate.

Macroscopically characteristics of acute pancreatic necrosis are areas of (1) grey white proteolytic destruction of parenchymal substance, (2) haemorrhage and (3) chalky white areas of fat necrosis. Characteristically, the peritoneal cavity contains a serous and slightly turbid, brown tinged fluid in which globules of oil

can be identified. (So called chicken broth fluid) Foci of fat necrosis may be found in fat depots of omentum, mesentry of bowel and properitoneal deposits. If the patients survives the acute necrotizing damage may resolve slowly and be replaced by diffuse (or) focal parenchymal (or) stromal fibrosis, calcification and irregular ductal dilatations occasionally liquefied areas are walled off by fibrous tissue to from small or large cystic spaces known as PSEUDOCYSTS.

The most common type of chronic pancreatitis is chronic calcifying pancreatitis mostly seen in alcoholics. All components of involved lobules are affected. There is atrophy of the acini, marked increase in interlobular fibrous tissue and a chronic inflammatory infiltrate around labules and ducts. The inter lobular and intralobular ducts are dilated and contain protein plugs in their lumina. The ductal epithelium may be atrophied (or) hyperplastic (or) may show squamous metaplasia. Grossly the gland is hard and exhibits foci of calcification and fully developed pancreatic calculi. Pseudocyst formation is common in this type of pancreatitis.

Another type is chronic obstructive pancreatitis. The distribution of lesion is not lobular and the ductal epithelium generally is less severely damaged. The most common cause of this type is stenosis of the sphincter of oddi, associated with cholelithiasis. The lesions are more prominent in the head of pancreas.

All Ca arise from ductal epithelium. Carcinoma head of the pancreas are fairly small lesions. Some lesions may be upto 8 to 10cm in diameter. The grey, white, scirrhous homogenous Tumour infiltrates and replaces the lobular architecture of a normal pancreas. Such lesions have poorly defined, infiltrative margins, extents to the margin of the duodemnum & CBD, Sometimes it produces either a small fungating lesion or an irregular ulceration. In this infiltrative growth, it surrounds and compresses the CBD (or) ampulla of vater causing biliary obstruction. Extension to portahepatic nodes are also common.

Ca of body & tail of pancreas are unusually large, hard and irregular mass. On cross section, it resembles the Ca head of pancreas but frequently extend more widely than those of the head. They invade the adjacent vertebral column, retroperitoneal space, spleen (or) adrenal, transverse colon & stomach Massive hepatic metabolism are quite common of Ca of tail and body of pancreas.

Histologicaly more or less they have well differentiated glandular patterns and are thus adenocarcinomas. The tumours may be mucinous or non mucin secreting. The glands are atypical, irregular, small and are usually lined by anaplastic cuboidal to columnar epithelial cells. About 10% either adenosquamous pattern (or) giant cell formation. 0.5% - Cystadenocarcinoma arise in cysts. Rarely acinar cell ca-arise form acinar cells.

Pancreatic cysts have been classified as True cysts and false cysts (Pseudocysts) True cysts contain epithelial lining where as false cysts do not have epithelial lining. Congenital cysts (or) true cysts usually multiple but occasionally occur as single. They range in size from microscopic lesions to large spaces upto 3 to 5cm in diameter. They are lined by smooth, glistening membrane lined by atrophied epithelial cells (or) low cuboidal epithelial cells. They are usually enclosed in their fibrous capsule and are filled with a clear to turbid mucoid (or) serous fluid.

Pseudocysts are collection of fluid that arises from loculation of inflammatory processes, necrosis (or) haemorrhages. These are clinically important and are always associated with pancreatitis secondary to alcohol abuse, biliary tree disease (or) trauma. These are usually solitary and measures 5 to 10cm in diameter. Cysts wall may be thin (or) thick and fibrous. They do not have an epithelial lining and have no connection (or) communication with surrounding ductal system. There may be a marked inflammatory reaction in the fibrous capsules. Cystic fluid is usually serous and turbid. But the pesudocysts of chronic pancreatitis are of retention type and the duct is decreased and strictured. Duct-cyst communication always present.

Cystic pancreatic neoplasms consist about 10%-20% of all cystic lesions. Depending on their serous (or) mucinous component they have been classified

into benign and malignant. Mucinouscystadenoma tend to progress to malignancy and serous cystsadenoma can be considered benign. Papillary cystic tumour, cystic isletcell tumour and acinar cystadenoarcinoma are other cystic neoplasms. Some may undergo necrosis and cystic degeneration, can present as a cystic mass and these include ductal adenocarcinoma, sarcoma & lymphomas.

Calcification of the rim of cyst wall is common in cystic tumour. Central ‘Sun Burst’ calcification is reported to be highly suggestive of serous cystadenoma and also occurs in 33% of mucinous adenocarcinoma.

MY STUDY

INTRODUCTION

I have studied 45 cases of pancreatic diseases. The breakup 45 pancreatic diseases is as follows.

Acute pancreatitis	–	12 cases
Pseudocysts of pacreatitis	–	7 cases
Chronic calculas pancreatitis	–	15 cases
Ca head of pancreas	–	10 cases
Ca body	–	1 case
Total	**–**	**45 cases**

The age and sex incidence of pancreatic disease is as follows.

Age	**Sex**	
	Male	**Female**
10-20	2	2
21-30	7	0

31-40	13	2
41-50	5	3
51-60	3	3
More than 60	2	3
	32	+ 13 = 45

CLINICAL PRESENTATION

ACUTC PANCREATITIS

Patient with acute pancreatitis mostly presented with pain abdomen (>70%). Pain acute in onset and presented with shock. Pain mostly in mid epigastric region and also some patients present with fever nausea, vomiting, and hiccup. Most of the patients were chronic alcoholic and less than 40 years old. On examination patient most patients were in shock.. There was tenderness in the mid epigastric region. Abdominal distension also present. Localized guarding, rigidity noted in epigastrium.

CHRONIC PANCREATITIS

Patient with chronic pancreatitis are mostly present with pain abdomen and also with pain referred to back and left & Right hypochondrium. Most are suffered form malabsorption and weight loss. Most patient are chronic alcoholic, On examination, tenderness over epigastic region present.

PSEUDOCYST OF PANCREAS

Patients with pseudocyst of pancreas are mostly present with pain abdomen, anorexia vomiting. One patient present with epigastric mass. 99% patient were chronic alcoholic, one patient developed pseudocysts of pancreas following a blunt injury abdomen and two patients developed pseudocysts of pancreas following an attack of acute pancreatitis. Patients with cysts in the pancreas due to chronic calculus pancreatitis are also present with abdominal pain, nausea, and vomiting.

CA HEAD OF PANCREAS

Patients with carcinoma head of pancreas are mostly present with jaundice, weight loss, anorexia, pain abdomen. One patient present with abdominal distension and engorged vein over anterior abdominal wall. One patient presented with progressive jaundice with severe anorexia. Two patients were chronic alcoholic and chronic smoker.

One patient presented with diabetes mellitus and another patient present. With pedal oedema and dyspnoea. On examination some patients have epigastric mass and 6 patients with hepatomegaly and GB palpable.

In my single case of Ca body of pancreas. The case was a female the main complaint was mild epigastric pain, anorexia. There was no jaundice.

Total No. of cases: 11

Symptoms	No of cases	Percentage
Jaundice	8	73%
Weight loss	9	82%
Pain Abdomen	6	55%
Anorexia	9	82%
Ascites	2	20%

INVESTIGATIONS

In my study all the patients presented with pain abdomen and with a history of chronic alcoholism were evaluated by the estimation of serum amylase level. On an average there was H/o alcohol consumption for 8 years with quantity about 200ml/day. It was found minimum elevation to very high level ranging from 180 U/L to 1544U/L.All the patient were evaluated for altered liver function tests. Routine blood investigations were also done.

RADIOLOGICAL INVESTIGATIONS

For all the patients with pain abdomen and chronic alcoholism and jaundice. Plain –X ray abdomen was taken. Six of the patients showed areas of calcification along the line of pancreas of varying size.

ULTRA SONOGRAPHIC EXAMINATION

1. Pseudocyst of pancreas

Most of the cysts were situtated in the head of pancreas. Sizes of the cysts were ranging from 4cm ×4cm to a maximum of 20cm ×15cm. This large cysts was found in a patient with history of blunt injury abdomen for that laparotomy has been done-2months back.

Ca Pancreas

In 11 patients of Ca pancreas some had mass in head of pancreas with liver enlargement and GB with thickened wall. They also showed dilatation of CBD and intra hepatic biliary radicals. In two patients liver secondaries with lymph nodal metastasis along greater curvature and pancreatic duodenal nodes also present. In one of the above two patients haemorrhagic ascities were also seen. In a single case of ca uncinate process of pancreas there was no infiltration of blood vessels.

CHRONIC CALCULUS PANCREATITIS

In chronic calculus pancreatitis patients. The ultra sonagraphic findings were pancreas with multiple large calculi packed in the entire length of the pancreatic duct of size varying from 1.5cm to 2cm diameter. Pancreatic duct was dilated. Gall bladder was distended with dilated intrahepatic biliary radicals.

ACUTE PANCREATITIS

In acute pancreatitis patients the ultra sonographic findings was oedematous pancreas. Areas of parenchymal necrosis were present.

CT SCAN

Ca Head of Pancreas

CT Scan finding of the Ca Head of pancreas was growth in the head of pancreas with intra hepatic and extra hepatic biliary radicals dilattion. No para aortic & porta hepatic nodal involvement. In two patients multiple liver secondaries and para aortic nodes were present.

Chronic Calculus Pancreatitis

CT scan findings of calculus pancreatitis was calculi in the pancreatic duct with dilatation of pancreatic duct. In one patient cystic degeneration of parenchyma was present. In one patient entire duct was studded with calculus & in one patient entire calcinosis of pancreas seen.

Pseudocyst of pancreas

CT scan findings of pseudocyst of pancreas was enlarged pancreas and cystic lesion of varying size mainly in the head of pancreas and some in the body and tail of pancreas.

Acute Pancreatitis

CT scan finding of acute pancreatitis was mostly haemorrhagic oedematous parenchyma and some areas of necrosis present.

Percutaneous Transhepatic Cholangiogram (PTC)

PTC finding of one patient was dilatation of intra hepatic biliary radicals and hepatic ducts with obstruction of common hepatic duct? malignant growth.

OGD finding of one patient was extra luminal compression present.

MANAGEMENT

ACUTE PANCREATITIS

Out of twelve cases of acute pancreatitis eleven patients were treated conservatively by the following regimen.

1. IV Fluids
2. Ryle’s tube aspiration
3. Analgesics
4. Antibiotics
5. H^2receptor antagonists
6. Sedatives

One of patients was treated surgically in the form of laparotomy followed by peritoned lavage with placement of bilateral flank drainage tubes, one in lesser sac and another in general peritoneal cavity.

PSEUDOCYST OF PANCREAS

Out of 7 cases of pseudopancreatic cysts, 4 cases has been treated conservatively by the following regimen, because of small size of cysts measuring < 5cm.

1. IV Fluids
2. Ryle’s tube aspiration
3. Analgesics
4. Antibiotics
5. H^2receptor antagonists
6. Anti cholinergic (Probanthine)
7. Sedatives

Three cases has been treated surgically. The different surgical procedures adapted were as follows

Cystogastrostomy for a cyst of size 20×15cm situated in the region of head of pancreas.

Cystojejunostomy for a cyst of size 15 ×15cm situated in the region of tail of pancreas occurring as a result of blunt injury abdomen. In this cases, 3 to 3.5 liters of necrotic material were aspirated.

External drainage of the cyst by a mallecot's catheter as it had reptured into peritoneal cavity.

CHRONIC CALCULUS PANCREATITIS

Out of 15 patients of chronic calculus pancretitis, one patient presented with psuedocyst with calculi in the pancreatic duct. This patient was treated surgically by pancreatic lithotomy followed by cystojejunostomy and jejunojejunostomy. 8 cases of chronic calculus pancreatitis without psuedocyst were treated surgically as follows.

- Pancreatico-lithotomy followed by pancreatico jejunostomy and jejunojejunostomy – six cases
- Pancreaticolithotomy followed by Roux-en-Y pancreatico jejunostomy and jejunojejunostomy – two cases.

Remaining 6 cases presented with entire calcification of pancreas and so treated conservatively for pain only.

CA HEAD OF PANCREAS

Out of 10 cases of Ca head of pancreas, six patients were treated conservatively. Four cases underwent radical surgical treatment. They were treated with WHIPPLE'S PROCEDURE – Pancreaticodeuodenectomy.

One case of Ca uncinate was treated by Whipple procedure.

For three cases, palliative surgical bye-pass procedure done.

For three cases, because of extensive infiltration only biopsy was taken. Post operatively they were treated by chemotheraphy.

For one patient chemotheraphy was given as per the ultrasonographic and CT scan finding of extensive liver secondaries and ascites.

DISCUSSION

In my study, I had come across 12 cases of acute panceratitis. All the patients were alcoholic except 2 persons who were female. I noticed that Alcohol is the prime etiological factor in acute pancreatitis. The other etiological factors are as follows:-

1. Alcoholism
2. Biliary tract disease
3. Trauma
    - Surgical
    - Blunt injury
    - Penetrating injury
    - ERCP
    - Aortography
4. Drugs
    - Thiazides
    - Steroids
    - Azathioprine
    - Fruscemide
    - Sulfonamides
    - Clonidine
    - Phenformin
    - Tetracycline
5. Metabolic disorders
    - Hyperparathyroidism
    - Hyperlipidaemis
    - Hypercalcaemia
6. Infections
    - Mumps
    - Coxsackie –B virus
    - Mycoplasma pneumonia
    - Infectious mononucleosis
    - Septicaemia
7. Congenital mechanical obstruction of pancreatic duct - pancreas divisum
8. Periampullary carcinoma

9. Hereditary pancreatitis
10. Vascular disease
    - Cardio pulmonary By pass
    - Polyarteritis nodosa
    - Athero embolism
11. Miscellaneous scarpion venom

In my study all the patients presented with pain abdomen (100%); vomiting and shock (50%). The other common symptoms were retching and hiccough rarely diarrhea, dyspnoea, cyanosis, haematemesis and malena may appear. In my study, except for epigastric tenderness and shock, no other signs were presented. Other signs were mild jaundice and abdominal distension in early stage. Other late signs were bluish discoloration of the skin around the periumbilical area (CULLEN'S SIGN) or in the loin (GREY TURNER'S SIGN), are noted around 4^{th} and 5^{th} day in cases of haemorrhagic pancreatitis but I didn't came across such signs in my study. Rarely polyarthritis (or) bone pain may be observed.

In my study, I came to know that serum amylase estimation is the diagnostic test for acute pancreatitis. The other tests which could be done are

1. Urinary amylase and lipase
2. Serum ribonuclease (or) deoxy ribonuclease
3. Hyperglycemia and glycosuria.
4. Hypocalcaemia

5. Methaemalbuminaemia
6. Blood coagulation tests (e.g) Serum fibrinogen level – elevated
7. Hyperilipidaemia
8. Elevated CRP
9. Urinary (TAP) test
10. Procalcitonin marker

Among the above sited tests serum lipase estimation is more specific for pancreatic disease than amylase. Because lipase is solely of pancreatic origin & Serum amylase level may be increased in intra abdominal pathologies like.

- Intestinal obstruction
- Perforated peptic ulcer
- Acute appendicitis
- Ruptured ectopic pregnancy

And extra abdominal pathological conditions like salivery gland disorders like

- Mumps
- Parotitis
- Renal failure
- Pneumonia
- Cerebral trauma

- Severe burns
- Diabetic ketoacidosis

Patients with acute pancreas may have normal amylase.

1. Urinary clearance of amylase ↑ shortly after pancreas inflammation. Here urinary amylase > 5000IU is diagnostic.
2. In hyperlipedemia there will be difficulty in interpretation
3. In chronic pancreatitis the pancreas is damaged to a certain extent that amylase can't be produced.

Amylase – creatinine clearance ratio is also a useful diagnostic tool in acute pancreatitis.

$$\frac{\text{Urine amylase}}{\text{Serum amylase}} \times \frac{\text{Serum creatinine}}{\text{Urine creatinine}} \times 100 = \text{the amylase – creatinine clearance ratio}$$

Normally this ratio is 1 to 4%. If it is greater than 6% it indicates acute pancreatitis.

In acute pancreatitis there may be ECG changes also. They are:

ST segment elevation (or) depression
Inversion of T waves
Extended T wave negatively
} (these are due to electrolyte imbalance)

In acute pancreatitis, plain X-ray abdomen and chest X-ray may show evidence of pneumoperitoneum. The radiological sings include intestinal distension in the region of the pancreas like

- Sentinel jejunal loop
- Colon cut-off
- Duodenal ileus.
- Generalized paralytic ileus
- Oblitation of psoas outline
- Elevation of left diaphragm
- Renal halo.

Ultrasonography abdomen may be used in the diagnosis of acute pancreatitis. Unfortunately the value of USG is often limited by the presence of air and fluid filled loops of bowel overlying and obscuring the pancreas.

Currently the most widely accepted and sensitive method used to confirm the diagnosis of acute pancreatitis is CT. That too dynamic CT (DCT) is the most sensitive index. CT findings in acute pancreatitis.

1. Pancreatic Changes
2. Parenchymal enlargement
3. Diffuse
4. Focal
5. Parenchymal oedema
6. Necrosis
7. Peripancreatic changes

8. Blurring of fat planes
9. Thickening of fascial planes
10. Presence of fluid collections
11. Non specific findings
12. Bowel distension
13. Pleural effusion
14. Mesenteric edema

CT SEVERITY INDEX BY BALTHAZAR ET AL

A : Normal pancreas consistent with mild pancreatitis

B : Focal or diffuse enlargement of gland, including contour irregularities and inhomogenous attenuation but without peripancreatic inflammation.

C : A & B plus peripancreatic inflammation.

D : C + single fluid collection

E : C + 2 or more fluid collection

Gas in pancreas (or) retroperitoneum

A – E compounds to 0-4 score

For necrosis

No	:	0	Score
< 1/3	:	2	
> 1/3	:	4	
> 1/2	:	6	

This can be scored upto 10points

Index	**Morbidity**	**Mortality**
0-3	8%	3%

4-6	35%	6%
7-10	98%	1.7%

The clinical course in upto 90% of patients with acute pancreatitis follows with mid self limited pattern. However in 10% to 15% of patients a severe form of illness may occur. It is possible to predict the severity of an attack of pancreatitis objectively by 11 early parameters identified by RANSON in 1974. Most useful in patients with pancreatitis not related to gall stones.

RANSONS CRITERIA:

On admission to hospital

1. Age greater than 55 years
2. WBC >16,000 cells/cumm
3. Fasting Blood glucose >200mgm/100ml
4. Serum LDH >350 IU/L
5. SGOT .250U/100ml

   With in initial 48 hrs of admission
6. Haematocrit fall >10% points
7. BUN elevation >5mgm /100ml
8. Serum calcium fall to <8mgm/100ml
9. Arterial Po^2<60mm of Hg
10. Base deficit >4m Eq/L

11. Estimated fluid sequestration >6L

In patients with less than 3 of these 11 signs, the mortality rate is 0.9%.

Less than 3 signs	–	0.9%
With 3-4 signs	–	18%
With 5-6 signs	–	50%
With >6 signs	–	90%

In my study, out of 12 cases of acute pancreatitis, 11 patients were treated conservatively by the following regimen,

1. IV fluids
2. Ryle's tube aspiration
3. Analgesics
4. Antibiotics
5. H^2 receptors antagonists
6. Anti cholinergic (Probanthine)
7. Sedatives

Proposed Non operative therapies for acute pancretitis are as follows Supportive measures

- IV fluid therapy
- Electrolyte
- Analgesics
- Nutritional support

- Antibiotics

- Respiratory support

Pancreatic exocrine secretion suppression

- Nasogastric suction
- Histamine H2 receptor antagonists
- Antacids
- Anti cholinergics
- Glucagons
- Calcitonin
- Somatostatin

Pancreatic enzyme inhibition

1. Protease inhibitors
2. Aprotinin
3. Fresh frozen plasma
4. Antifibrinolytics
5. Chloroquine
6. Phospholipase A inhibitors

Pancreatic protection from oxygen derived free radicals

1. Free radical scavengers
2. Xanthine oxidase inhibitors
3. Isovolemic haemodilution

Elimination of toxic intraperitoneal compound

- Peritoneal dialysis

One patient of acute pancreatitis was treated surgically in the from of laparotony followed by peritioneal lavage with placement of bilateral flank drainage tube.

There are four situations when operative intervention is indicated in a acute pancreatitis patients

1. When the diagnosis is in doubt
2. Patients with known biliary stone disease
3. Failure of patient to improve on medical management
4. Treatment of secondary pancreatic infections like, pseudocyst formation. Abscess formation, haemorrhage resulting from pseudoaneurysm (or) sectorial (left sided) portal Hypertension.

When laprotomy is performed early in the course of acute pancreatitis, one or more of the following procedures may be advisable.

1. Laparotomy alone
2. Placement of catheters for peritoneal lavage.
3. Biliary decompression via a cholecystostomy (or) a T-Tube in the CBD
4. Operative Cholangiogram
5. Cholecystectomy, common bile duct exploration and choledocholithotomy with (or) without a sphincteroplasty.
6. Total (or) sub total pancreatic resectomy
7. Pancreatic and retroperitoneal debridement and drainage

8. Decompression gastrostomy and feeding jejunostomy.

Other modalities are

- Ultrasound guided drainage.
- Endoscopic cystogastrostomy

Two surgical pitfalls in acute pancreatitis are

1. To operate too early and do too much
2. To operate too late and do too little

CHRONIC PANCREATITIS

In my study, I have come across 15 cases of chronic pancreatitis of which 8 cases are of obstructive type with cyst and 6 cases are non obstructive type. The course of obstruction of pancreatic duct in my study was mainly due to calculi.

They other causes of obstruction are

1. Congential (or) Acquired of the pancreatic duct.
2. Pancreas divisum
3. Duct obstruction from tumours
4. Inflammation of the papilla of vater
5. Protein malnutrition
6. Cystic fibrosis
7. Hypercalcaemic states

Chronic pancreatitis can occur as a genetic condition transmitted as a Mendelian dominant trait. The condition is rare.

20 to 30% of chronic pancreatitis has no apparent cause so termed as idiopathic pancreatitis.

In my study, 75% of the cases of chronic pancreatitis was caused by alcoholism. Alcoholic pancreatitis generally occurs in patients who consume alcohol for at least 2 years and usually between 6 to 10 years.

Alcohol causes pancreatitis by following

1. Any one of the mechanism by inducing spasm of the sphincter of oddi thereby creating an obstruction to the outflow of pancreatic juice.

2. Alcohol is also a cellular metabolic poison and it has deleterious effects on the synthesis and secretion of digestive enzymes by the pancreatic acinar cells. This causes an increase in the concentration of enzyme protein in pancreatic juice, and the eventual preciptitation of this protein in the pancreatitis ducts. Calcium may also precipitate with the matrix protein plugs and obstruct the pancreatic ducts.

3. Alcohol increases the permeability of pancreatic duct there by initiate enzymatic leakage. This may cause pancreatic injury.

4. Alcohol significantly decreases pancreatic blood flow several hours after ingestion this may cause ischemic injury to gland.

In my study, most of the chronic pancreatitis patients had principal symptom of abdominal pain radiating to back or to left. Most of the patients were emaciated. Most of them were chronic alcoholic some were with diabetes mellitus. Repeated pain attack is characteristic of chronic pancreatitis. Pain free

intervals becomes shorter and the pain eventually occurs everyday. Pain is mainly due to increased intraductal pressure upto 30 to 50cm of H_2O. (Normal upto 20cm of H_2O). Eating may increase the pain, so many patients avoid food and lose weight. Significant exocrine insufficiency will occur if 90% of secretory capacity of pancreas is lost. The major consequences are steatorrhoea and creatorrhoea. They may complain of bulk, offensive, fatty and oily stools.

Potential cause of pain is chronic pancreatitis

1. Pancreatic ductal hypertension
2. Inflammation of intrapancreatic nerves
3. Loss of protective perineural sheath in pancreatic nerves
4. Pancreatic ischemia
5. Pseudocyst
6. Pancreatic & peripancreatic inflammation
7. Cholangitis.

Early in the disease serum amylase and lipase concentrations are elevated. As the disease becomes advanced, they often remain normal. Mild elevations of serum bilirubin alkaline phosphatase and SGOT and mild depression of serum albumin can occur. Pancreatic function test are rarely indicated in chronic pancreatitis patients. ERCP and CT scan are more useful diagnostic stools.

In 30 to 50% of patients with chronic pancreatitis plain X-ray abdomen reveal pancreatic calcifications.ERCP study provides important information about “ductal anatomy” that may influence a decision for surgery.e.g. for a dilated duct patient pancreatico jujunostomy is indicated. For a normal duct pancreatic resection is advisible. Strictures, cysts and ductal calculi may be seen. The characteristic “chain of lakes” seen. CT SCAN also useful for ductal anatomy Billary dilation and the level of bile duct obstruction in defined clearly. It also provides the most precise information about the size and configuration of the pancreas.

More than 2/3rd of chronic pancreatitis patients have diabetes mellitus. But the DM is usually mild and rarely associated with ketoacidosis and vascular complications.

Non operative management of chronic pancreatitis are

1. Control of abdominal pain
2. Treatment of endocrine insufficiency
3. Treatment of exocrine insufficiency

Control of abdominal pain

- Advise to stop alcohol intake (about 50% of patients has some pain relief when they stopped alcohol)
- Advise to consume semi solid or liquid diets instead of solids.

- Carbohydrate more
- Fat & protein less
- H_2 receptor antagonists
- Oral pancreatic enzyme supplements.
- Parentral somatostatin analogue to inhibit pancreatic secretion e.g.octreortide
- Attempts to control pain often require early use of non narcotic analgesics followed later by narcotic analgesics.

Treatment of exocrine insufficiency

1. Diatary restriction of fat is important.
2. Pancreatic enzyme replacement e.g. cotazym, ilozyme, viokase
3. H2 receptor blocking agent

Treatment of endocrine insufficiency

1. Mild elevations of blood sugar do not require treatment fasting level >250 mgm/dl should be managed with insulin.
2. Maintain the fasting level around 200 mgm/dl

Surgical Treatment

In this study, out of 15 patients of chronic calculus pancreatitis, one patient presented with pseudocyst with calculi in the pancreatic duct. The operative procedure was pancreatico lithotomy followed by cystojejunostomy

and jejuno jejunostomy. Another 8 patients were treated surgically. The different procedures are as follows.

- Pancreaticolithotomy followed by pancreaticojejunostomy and jejunojejunostomy – Six cases.
- Pancreaticolithotomy followed y Roux-en-Y pancreaticojejunostomy and jejunojejunostomy – Two cases.

The primary goal of operative management is relief of pain, the secondary consideration is to preserve maximal endocrine and exocrine function. prior to surgical intervention ERCP &CT study is must to study the ductal anatomy

1. Drainage procedure – for dilated duct
2. Pancreatic resection – for Normal (or) Narrow duct

Drainage procedure (pancreatico jejunostomy)

The main pancreatic duct has a normal diameter of 4 to 5 mm in the head, 3 to 4 mm in the body and 2 to 3mm in the tail. If the diameter is more than 7 to 8 mm in body and head, a pancreatico jejunostomy (puestow procedure) is technically feasible with adequate stroma length is 6-10cm.

1. Operative mortality is about 4%
2. Drain will close spontaneously
3. Patient may gain weight
4. Pain relieved in about 80 to 85% patients
5. Sometimes stenosis of pancreatico jejunal anastomosis may occur, then pancreatic resection is advisable.

Pancreatic resections

1. Pancreatico duodenectomy
2. Pylorus preserving pancreatico duodenectomy
3. Pancreatic Head resection
4. Distal pancreatectomy

The main indication of pancreatic resection for a pancreatitis patient is

1. To relief pain
2. Failure of drainage procedure
3. When the pathological changes involve one part of the gland and the rest is less diseased.
4. When the diagnosis between chronic pancreatitis and pancreatic cancer is in doubt

Pylorus preserving pancreatico duodenectomy

- The entire stomach, Pylours and first 3 to 4cm of duodenum are preserved
- Technically easier and more quicker
- Preserve gastic function

PANCREATIC HEAD RESECTION

Indications

- Most of pathological changes involving
- The head of pancreas
- ½ of duodenal compression
- 2/3rd of CBD compression
- 1/4th of portal vein compression

Resected Structures

- Head of the pancreas is resected

- Entire stomach and duodenum preserved.
- Body & tail of pancreas as well as a thin rim of pancreatic tissue in the 'C' loop of duodenum also remain

Theoretical advantages

- Preservation of gastroduodenal and biliary continuity and function
- Prevent the development of Diabetes

DISTAL PANCREATECTOMY

Indications

1. Recurrent episodes of pancreatitis with multiple pseudocysts in the tail of the pancreas and splenic vein thrombosis
2. Recurrent episodes of pancreatitis with a stricture in the main duct in the body of the pancreas and a dilated duct.

Resected Structures

1. Variable amounts of tail (or) body of the pancreas are resected
2. Spleen is removed in most instances
3. Head of the gland is preserved

Complications:

1. Pancreatico jejunal fistula
2. Choledocho jejunal fistula
3. Injury to CBD

   Pain relieved in about 85% after resection. About 60 to 70% after drainage procedure

The principle cause of deaths are:

- Upper respiratory malignancies
- Malnutrition
- Complications of diabetes

PSEUDOCYST OF PANCREAS

In my study, out of 7 cases of pseudocyst of pancreas, all the patients were chronic alcoholic. They presented with abdominal pain, vomiting and upper abdomen mass two patients developed pseudocyst following a blunt injury abdomen and one patient developed following an attack of acute pancreatitis. Two patients presented with enlarged GB with signs of obstructive jaundice.

ETIOLOGICAL FACTORS OF PSEUDOCYST ARE SHOWN AS FOLLOWS

Etiological factors of pseudocyst

Alcoholism	-	4
Gall stones	-	Nil
Trauma	-	2
Idiopathic	-	1

All the patients had raised serum amylase level. Ranging from 426 to 910 U/L Few patients show elevated alkaline phosphatase and serum bilirubin and elevated SGOT and SGPT levels. CT scan was useful in assessing the age of the cyst. ERCP can define the pancreatic ductal anatomy and cyst-duct communication. In some patients OGD was also done, OGD showed extra

luminal impression of the stomach due to a lesion situated posterior to the stomach.

In my study the course of pancreatic pseudocyt was analysed. About 30% of fluid collections disappear spontaneously. Smaller cysts of size 4 to 5cm resolved spontaneously with conservative line of management only few patients with large size cyst treated surgically. In this study in one patient cystogastrostomy and in another patient cystojejunostomy was done. These entire pseudopancreatic cysts were present in the region of Head of pancreas. Most were single and in only one patient multiple small cysts occurred. One case was treated by ultrasound guided percutaneous drainage.

Surgical treatment depends on the size, duration of the cyst, maturity of the cyst wall and presence (or) absence of infection of cyst contents.

Large adherent retrogastric cysts were drained into the stomach, cysts with the duodenal sweep were drained into the duodenal and in the tail of pancreas were drained into jejunum.

Complication of the internal drainage include

- Gastro intestinal Haemorrhage
- Cyst recurrence
- Sepsis

Complications are more frequent following cystogastrostomy than cystojenostomy. Cystogastrostomy is associated with shorter operative time and hospital stay, but a higher incidence of cyst recurrence.

The optimal therapeutic approach for multiple pseudocysts is internal drainage. The options are internal cystostomy combined with enteric drainage for contiguous cyst and drainage by multiple Roux-en-Y cystojejunostomies for non-contiguous cysts.

In this study external drainage of the pseudo pancreatic cyst was done in a patient. Since it had ruptured into the peritoneal cavity. External drainage is the operation of choice for

1. Infected cysts
2. Those associated with haemorrhage
3. Free rupture into the peritoneal cavity
4. Immature cysts that will not hold sutures.

It will be associated with a persistent pancreatic fistula, recurrence and mortality.

There are many non surgical techniques for treating the pseudo pancreatic cysts.

They are

1. Needle aspiration
2. Percutaneous catheter drainage

3. Percutaneous cystogastrostomy
4. Endoscopic cyst-enteric drainage
5. Endodcopic transpapilary approach.

PERCUTANEOUS CATHETER DRAINAGE

Percutaneous catheter drainage is an excellent, initial option in patients:

1. Who are critically ill
2. Who are high surgical risks
3. Who have infected pseudocysts

Success rate is about 93%.Morbidity is about 13%

The drawbacks include

1. Occurrence of controlled external pancreatic fistula.
2. Risk of drain site infection
3. High incidence of cyst persistence (or) recurrence

Cystogastric Drainage

Placement of a double pig tail stent using fluroscopy with ultra sound guidance technique is an easy and safe alternative. Surgical drainage stents can be placed within a few weeks of development of the cyst and have been kept for a period ranging from 6 weeks -10 months without any problems.

Endoscopic drainage is feasible and safe for selected cases of pancreatic pseudocyst. The success of this procedure relies on close proximity of the cyst to bowel wall and the choice of drainage site. It can be made only by endoscopic

demonstration of an obvious intra luminal bulge. This technique should not be used if the wall thickness is greater than 10mm

The serious complications are

1. Arterial bleeding
2. Infections
3. Duodenal perforation

Rupture of the pseudocyst into the neighbouring bowel is increasingly reported. Perforation into the colon carries a high morbidity and mortality where as fistula into the stomach (or) small bowel result in uneventful recovery.

Haemorrhage is an uncommon but serious complication of pancreatic pseudocysts and the source of bleeding may be rupture of a pseudo aneurysm inside the pseudocyst. Vascular involvement can be diagnosed on Ultrasonogram and angiography. Angiography embolization, suture ligation of the bleeding vessel are the two lines of management.

Several factors need to be considered in the choice of a suitable surgical method.

1. Site
2. The number of cyst
3. Pancreatic endocrine and exocrine capacity
4. Mechanical obstruction of the biliary tract gastric outlet
5. Status of the main pancreatic duct

Resection indicated when the patient has severe pancreatic endocrine insufficiency and when multiple pseudocysts are located in the head of pancreas which are not suitable for internal drainage

Ca Head of Pancreas

In this study I have come across 10 cases of Ca head of pancreas. Most of the patients had complaints of weight loss (90%), anorexia (90%), jaundice (77%), and pain abdomen (60%). One patient with abdominal ditension. Two patients were chronic alcoholic and chronic smoker. One patient with Diabetes.

Pancreatic ductal adenocarcinoma is about 90% of all malignant neoplasm of the gland. Highly fatal disease, 5 year survival rate is 1-2% only accounts for 10% all the cancer of digestive tract.

- Fourth most common cancer of all sites as a cause of death (behind lung, colorectal and breast)
- More common in older people (Sixth to Eighth decade)
- Commoner in men than in women

Pain and weight loss are the two main consistent symptoms. It may be episodic and related to meals it may become constant and chronic. Weight loss is severe and rapid. Haematemesis and malena are late features. Migratory

Thrombophlebitis (Trousseau's sign) can be present. Other signs are painless progressive jaundice and clay coloured stools.

I have come across Ca uncinate pancreas in one of the patient.

On examination epigastric mass may be felt liver & GB may be palpable (30%). Distant metastasis in the supraclavicular fossa (Troisier's sign) may present Ascites may be present.

Etiopathogenesis

Common in age 60-80. Common in males

RISK FACTORS

A. Demographic Factors

- Age
- Black race
- Male
- Gewish race

B. Host factor

Certain genetic syndromes are

- HNPCC
- BRCA – 2
- Peutzjehar syndrome
- Ataxia telangectacia
- Familial atypical multiple melanoma mole syndrome
- Hereditary pancreatitis
- Diabetes

- Cystic fibrosis
- Chronic pancreatitis

C. Environmental factors

- Cigarette smoking
- Diet rich in fat (or) meat

D. Predisposing medical condition

- Following gastrectomy – increased nitrosamines
- Following cholecystectomy – increased CCK

E. Occupational

- Leather tanning, textiles, chemical like chlorhydrin, halogenated hydrocarbon

Three patients came to hospital in very late stage of cancer. The causes were:

1. Asymptomatic tumour
2. Patient delay
3. Financial and personal reasons
4. The patient may not have ready and easy access to competent diagnostic centres.

All the patients presented with liver function test abnormalities, including increased levels of total bilirubin, alkaline phosphatase and transaminases.

Because of obstructive nature of these tumours, alkaline phosphatase is generally more elevated than the transaminases.

Tumour associated antigen, CA 19-9 and CA 494 has a sensitivity and specificity for pancreatic cancers. Other marker SPAN-1, CA-50.

Ultrasonography and CT scan are more useful tests. Both test confirm the obstructive nature of jaundice by demonstrating dilated intra hepatic and extra hepatic bile ducts.

CT is more useful than USG in determining the level of obstruction, demonstrating the presence of pancreatic mass (as small as 1 cm in size) and detecting liver metastasis or local vascular invasion MRI has no apparent advantage over CT. Helical spiral CT is the imaging modality of choice for diagnosis the staging of Ca pancreas.

ERCP (Endoscopic retro grade cholangio pancreatography) may be important if the differential diagnosis includes chronic pancreatitis.

Endoscopic examination is useful for visualization of ampullary and duodenal carcinoma and for biopsy taking.

The percutaneous approach is usually technically easier with a dilated biliary tree and offers the advantages of defining the proximal biliary system that will be used in reconstruction.

Duodenal drainage studies are recommended to obtain materials for cytological examination only. When ERCP has failed for technical reasons biliary drainage is useful in selected patients with advanced malnurtrition, sepsis and/or correctable medical conditions.

Percutaneous pancreas biopsy for surgically unresectable condition to get tissue diagnosis for chemotherapy.

In my study all the patients with Ca pancreas are kept in a good state of nutrition and hydration with supplemental IV fluids, elemental diet and multivitamin as deemed necessary. Blood clotting deficiencies are corrected by giving Vit K for 3 days daily.

Eventhough the bilirubin levels of these patients were elevated to a maximum level of 15.8mgm% we didn't performed biliary decompression procedures as cholecystostomy (or) T-Tube drainage of CBD preoperatively. But ideally speaking for any patient with raised serum bilirubin level more than 12mgm%, biliary decompression procedures either through transhepatic route (or) endoscopic approach should be done.

In this study, we selected the patients for radical treatment only when they found fit, i.e, not associated with distant metastasis, ascites, too old age.

In this study we did Whipple's operation (pancreatico duodenctomy) in four patients. In whipple's surgery we removed, head and neck of pancreas

together with duodenum, distal half of stomach, lower CBD, Gall bladder and upper jejunum and as much of regional lymph nodes as possible. Followed by triple anastamosis choledocojejunostomy, pancreaticojejunostomy, gastrojejunostomy,

The other surgical options are

1. Pylorus preserving surgery
2. Total pancreatectomy
3. Regional pancreeatectomy

In total pancreatectomy, along with contents of whipple's operation, the spleen, body and tail and regional lymph nodes are removed.

In Regional pancreatectomy along with the contents of total pancreatectomy, the transpancreatic portion of the portal vein, celiac axis, superior mesenteric artery and middle colic vessels are removed.

Among the three surgical options, total pancreatectomy with regional lymphadenectomy is recommended for the following reasons:

1. Pancreatic cancers are potentially multifocal in origin
2. Gross and histological tumour spread have been documented at the line of resection.
3. Malignant viable cells are often present in the obstructed pancreatic ductal system and if the gland is divided, this may be a source of seeding for local recurrence.

4. The existence of lymphatic exchange between the head and the body of the pancreas has been amply demonstrated.
5. Excision of the whole pancreas eliminates the risk of post operative pancreatitis.
6. Preservation of endocrine (or) exocrine tissue is not sufficient justification for leaving part of the pancreas in situ. Over 80% of all pancreatic cancer patients are diabetic at the time of presentation.

A cancer of the pancreas is considered unreasectable if there are:

1. Distant metastasis (liver or peritoneal)
2. Invasion of major vessels (portal vein, hepatic artery, superior mesenteric vessels and celiac artery)
3. Any extension beyond the area of usual total pancreatectomy specimen. Puckering of the transverse mesocolon per se does not always indicate unresectability. It can be removed along with total pancreatectomy specimens.

In my study post operatively, two patients developed renal failure symptoms and hypoproteinemia. Hypoproteinemia has been corrected by administration of injection Astymin and human albumin and fresh blood. Patients with renal failure symptoms were treated with diuretics as per the advise given by the nephrologists.

The other expected complications are haemorrhage, sepsis, mesenteric thrombosis, liver insufficiency, myocardial infarction, cerebrovascular accident, congestive heart failure and pulmonary embolism. The incidence of haemorrhage

is reduced by meticulous pre operative preparation and adequate replacement of blood and clotting factors during operation.

The re operation is indicated if:

1. If there is a reason to suspect a major bleeding site.
2. When clot accumulation in the abdomen causes distension and tamponade.
3. When a consumption coagulopathy is recognized.
4. In one patient, he developed post operative biliary leak. Patient was treated conservatively. Leakage from the biliary – enteric anastomosis or from the gastrojejunostomy are largely preventable by careful and proper construction of anastomosis. Complications that are usually non fatal include pneumonitis, gastric retention, paralytic ileus, bowel obstruction, wound injection, wound deheisence, atrial fibrillation, faecal fistula and gastrojejunal fistula.

Post operatively the jaundice had come down. Persistence of jaundice due to small bowel obstruction. The obstruction may be due to recurrent tumour and simply due to adhesions. Laparotomy may be indicated to establish the diagnosis and to relieve the obstruction.

MONITORING OF RECURRENCE

To assess the recurrence of tumour some tumour markers are more useful. They are:

1. Pancreatic oncofetal antigen (POA)
2. Carbohydrate antigen CA 19-9
3. Carcino embryonic antigen (CEA)

Serial monitoring of either marker may be useful in confirming the completeness of surgical excision and in the detection of recurrent pancreatic cancer.

Adequate pancreatin tablets (Viollase, pancrease) must be taken with each meal. The patient is advised to take a low fat diet in the form of frequent regular small meals.

The mortality rate in my study is 20%. One death is due to renal failure, and occurs after one month. Death is usually due to metastatic pancreatic cancer and another death occurs in first POD in my case due to consumption coagulopathy.

PLACE OF THE WHIPPLE'S OPERATION

If the surgeon can not be sure of the exact site of origin of the tumour at operation, he will do standard whipple operation and immediate careful examination of the specimen by the pathologist and surgeon. If the tumour is not originating from the pancreas, the operation is adequate. If the surgeon and pathologist is not sure about the origin of the tumour, convert the procedure into total pancreatectomy.

In my study we have done palliative surgical procedures for three patients. The palliative procedure are done to relieve jaundice, pruritis, impending cholangitis and for relief of duodenal obstruction.

Anterior gastrojejunostomy with jejunojejunostomy was done to relieve duodenal obstruction.

Cholecystojejunostomy with hepaticojejunostomy to relieve jaundice.

In this study we have taken biopsy from un resectable tumour of pancreas in 3 patients and histopathological examination done. After improving the general condition of the patient we gave chemotherapy.

When the patient is unfit or refuses operation, an alternative method of palliating the obstructive jaundice is by endoscopic sphinceterotomy and placement of a biliary stent.

In general the palliative surgeries are divided into operative and nonoperative.

OPERATIVE

- They are indicated to relieve biliary obstruction
- To avoid or treat duodenal obstruction
- Palliative tumor associated pain

For biliary obstruction – hepaticojejunostomy or choledocojejunostomy

For duodenal obstruction – retrocolicgastrojejunostomy

For pain

- Celiac ganglion block with 50ml of 50% alcohol (or) with 20ml of 6% phenol.

- Cordotomy, extensive sympathectomy and stereotactic thalamotomy have all been tried.

NON OPERATIVE

- Endoscopic stenting
- Percutaneous transhepatic biliary drainage

Chemotherapy

- 5 – FU
- Gemcitabine
- Improvement in median survival
- Improvement in pain control
- Potent radio sensitizer

Hormonal therapy

- Estrogen, androgen may affect pancreatic growth
- Two hormones CCK, gastrin has been under trial

Gene therapy

- Cytokine secreting pancreatic adenocarcinoma, vaccine is under trial.

CONCLUSION

1. The incidence of pancreatic disease in this hospital is not very low.
2. Male :Female ratio =2.5:1.
3. The most common pancreatic disease in this institution is chronic pancreatitis of pancreas. The incidence is 33%. Other diseases:

   Acute pancreatitis-27%

   Pseudocyst – 15.3%

   Pancreatic cancer -26%
4. Chronic alcoholism is the most common etiological factor for all pancreatic disease.
5. In case of chronic pancreatitis pancreatico-jejunostomy has better results.
6. For Ca Head of pancreas, whippli's procedure carries 20% mortality rate with average survival rate of 6 months.
7. In case of pseudocyst of pancreas for supracolic compartment cysto gastrostomy and for infracolic compartment cystojejunostomy has better results.
8. Survival rate is better in younger age group compare to older.

BIBLIOGRAPHY

1. Burns G.P. and Bank .S (eds).(1992) Disorders of the pancreas, McGraw Hill New York
2. Carter, D.C and Warshaw, A.L.(eds.) (1989) Pancreatitis, Churchill Livingstone, Edinburgh
3. Fraker D.L. and Norton, J.A.(1989) Gastroenterology clinics of North America, 18,805-29
4. Moosa, A.R.(eds.)(1980) Tumours of the pancreas, Williams and Wilkins, Baltimore
5. Moosa A.R.(eds.)(1984), Diagnostic test and procedures in acute pancreatitis, New England Journal of Medicine, 311,639-43
6. Moosa, A.R. Robson , M.C.and Schimpff S,C (1992). A comprehensive Text book of Oncology, Williams and Williams and Wilkins, Baltimore
7. Mozell, E., Stenzel .P, Woltering, E.A., Rosch .J, etal. (1990) Current problems in surgery, 27,309-86
8. Ranson, J.H.C. (1985) Acute pancreatitis in current Problems in Surgery Year Book Chicago
9. Schwartz, S.I., and Ellis, H (eds) (1985) Maingot's Abdominal operations. Appleton – century-Crofts, New York.
10. Warshaw, A.L.(eds) (1984) Progress Symposium on pancreatic cancer, World journal of Surgery 8 (6)

11. Cooperman, A..M. and Hoerr, S.O.(eds) (1978) Surgery of pancreas, C.V.Mosly,
12. St. Louis
13. Trede, M. and Carter, D.C (eds) (1993) Surgery of the pancereas, Churchill Livingstone, Edinburgh.

Govt. Rajaji Hospital

Madurai Medical college, Madurai-20

PROFORMA FOR A STUDY OF PANCREATIC DISEASES

Name : Occupation :

Age : Date of Admission :

Sex : Date of Discharge :

CLINICAL PRESENTATION

H/o. Jaundice-Progressive /Non Progressive

H/o. Pain abdomen

H/o. Alcoholism

H/o. Injury

H/o. Vomiting

H/o. Anorexia

H/o. Weight less

H/o. Mass

INVESTIGATIONS

Urine -Bile Salts Haemoglobin Heamatocrit

Bile pigments Blood grouping

Blood -Urea BT

-Sugar CT

Serum Creatinine

Serum amylase

LFT

Serum bilirubin :

Serum Proteins :

SGOT :

SGPT :

Alkaline Phosphatase :

RADIOLOGICAL INVESTIGATIONS

Plain X-ray abdomen :

Barium Meal Series :

USG-Abdomen :

CT Scan :

OGD :

PTC :

ERCP :

Operative Findings :

Procedure Done :

Post Operative Period :

Complication and its

Management :

CONTENTS

Acknowledgement

It gives me immense pleasure to express my deep sense of gratitude to my chief Prof. Dr. V.Narayanasamy , M.S., for his able guidance continous supervision. Constant help and encouragement during the course of my study and preparation of this dissertation.

I take up this opportunity to express my sincere gratitude to Prof. S.Koilpillai Packiaraj, M.S., Professor and Head of Department of Surgery his co-operations and encouragement during the course of my study.

I wish to express my thanks to the Dean Dr.J.R.Vijayalakshmi , M.D., Madurai Medial College, Govt. Rajaji Hospital, Madurai for permitting me to use the clinical materials of this hospital for the study.

I wish to express my gratitude to my senior Professors and Assistant Professors who helped me to complete this work.

Last but not least, I am grateful to all the patients without whom this work would not have been completed.

Madurai

Date: